The association of BMI with peak heart rate and peak oxygen pulse in apparently healthy adults

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"ONDERGETEKENDE

Matthijs van Harten,

bevestigt hierbij dat de onderhavige verhandeling mag worden geraadpleegd en vrij mag worden gefotokopieerd. Bij het citeren moet steeds de titel en de auteur van de verhandeling worden vermeld."

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ABSTRACT

The association of BMI with peak heart rate and peak oxygen pulse in apparently healthy adults

Background: improving cardiorespiratory fitness (CRF) is important in obese individuals. To guide physical training to improve CRF, knowledge of cardiac response to exercise, including peak heart rate (HR_{peak}) and peak oxygen pulse, is crucial because optimal training intensity is routinely determined as a percentage of HR_{peak}. Cardiac response is reported to differ between obese and normal weight individuals. The association of body mass index (BMI) with HR_{peak} and peak oxygen pulse is not well understood.
Aims: primary aim was to determine the association of BMI with HR_{peak} and peak oxygen pulse in apparently healthy individuals in various BMI subgroups. Secondary aim was to explore the role of CRF in the association of BMI with HR_{peak}.
Methods: a cross-sectional observational study was executed. Existing data of cardiopulmonary exercise testing (CPET), originating from The Low-Lands Fitness Registry, were used to analyze the association of BMI with HR_{peak} and peak oxygen pulse with linear regression analyses. Further analyses in BMI subgroups were then performed to compare differences in associations. The role of CRF was analyzed by introducing this variable into the regression analyses.
Results: data of 7990 participants were included. Median age was 31.7 and 5.2% was obese. BMI was significantly associated with HR_{peak} in the total sample, but not in separate BMI subgroups. Based on studied interactions, the association of BMI with HR_{peak} was different in obese individuals compared to non-obese. BMI was also significantly associated with peak oxygen pulse in the total sample and in some of the BMI subgroups. Partial R^2 of BMI in the models ranged from 0.000 – 0.033. CRF was significantly associated with HR_{peak} in the total sample and all BMI subgroups.
Conclusion: BMI is associated with HR_{peak} and peak oxygen pulse and these associations differ between overweight and obese individuals and individuals with a normal body weight. CRF plays an intervening role in the association of BMI with HR_{peak}.
Clinical relevance: the impact of BMI on HR_{peak} and peak oxygen pulse is limited and may not be relevant for clinical decision-making.

Keywords: body mass index; obesity; heart rate, peak; peak oxygens pulse; cardiorespiratory fitness.

INTRODUCTION

Obesity is a global health problem with a prevalence of 650 million in 2016.[1] The prevalence nearly doubled in the period from 1980 to 2008[2] and the problem is expected to grow further, with a predicted increase of obesity prevalence of 33% in 2030[3]. Obesity in adults is defined as a body mass index (BMI) of 30 kg $\cdot m^{-2}$ or more[4] and is associated with a range of health problems, like osteoarthritis, diabetes type 2 and cancer[5,6]. Moreover, obesity is associated with forms of cardiovascular disease (CVD), like heart failure, hypertension and coronary heart disease.[7] Higher risk for CVD was also reported for overweight individuals (BMI 25 – 29.9 kg $\cdot m^{-2}$).[8] Low cardiorespiratory fitness (CRF) is found to be an intervening factor in the association of obesity with the prevalence of CVD and with mortality in individuals with CVD.[9–11] Furthermore, a low level of CRF is a strong predictor itself for CVD.[12,13] Therefore, improving CRF - usually measured by peak oxygen uptake (VO_{2peak})[12] - is important in the obese population. Aerobic training can reduce obesity and increase CRF.[14] For effective training, cardiac response to exercise like peak heart rate (HR_{peak}) can be used to determine optimal training intensity.[15,16] HR_{peak} and CRF can be determined with cardiopulmonary exercise testing (CPET).[17]

In the obese population, cardiac response to exercise differs from the non-obese with consequences for the use of parameters like HR_{peak}.[18] For optimal training prescription in overweight and obese individuals knowledge of changes in cardiac function during (maximal) exercise in these groups is important.[19] Several studies[20–25] have investigated the association of BMI with HR_{peak}. In a cross-sectional study with 3320 participants, a difference of 2.3 beats per minute ($P < 0.001$) of HR_{peak} was found in obese persons compared to individuals with a healthy body weight.[21] Tanaka et al.[25] did not find other variables than age to be associated with HR_{peak} though, whereas Zhu et al.[24] found an increase in BMI to be inversely related to HR_{peak}. In this study, individuals with a BMI of 26 kg $\cdot m^{-2}$ or higher had the lowest HR_{peak}, but no distinction was made for the magnitude of overweight and obesity eventually. In summary, predictive models of HR_{peak} seem inaccurate and there is controversy about the association of other variables than age with HR_{peak}, including BMI.[20–26]

In addition to this uncertainty, the validity of HR_{peak} estimations in an obese population can in particular be questioned. Several differences in cardiac function have been reported in obese individuals when compared to non-obese individuals.[27] Cardiac output was reported to be higher for obese people. This was mainly the result of a larger stroke volume.[7] Heart rate in rest is higher on average and heart rate variability lower with obesity.[28] Furthermore, obesity is associated with left ventricular hypertrophy, ventricular diastolic dysfunction and fatty infiltration of the right ventricle.[29] Left ventricular diastolic dimension is related to HR_{peak}: the larger the heart, the lower HR_{peak}.[30] During exercise, a larger stroke volume and cardiac output is present in obesity and lower arteriovenous oxygen difference and ejection fraction during exercise have been reported, when compared to normal-weight peers.[31] Stroke volume measurement is rarely applied during CPET and therefore surrogates are often being

used. Peak oxygen pulse is a surrogate of stroke volume and can be used a predictor of cardiac mortality for people with chronic heart failure and is therefore important for risk stratification.[32,33] Peak oxygen pulse (maximal amount of oxygen taken up per heart beat) is determined by the ratio of the variables VO_{2peak} and heart rate. Both these variables tend to behave differently in people with obesity.[34]

Several studies investigated cardiac function during exercise in the overweight and obese.[14,19,31,34–36] Small samples were used however, and the association of BMI with HR_{peak} and peak oxygen pulse remained mostly unattended. Better insight into the association of BMI with these parameters is warranted, especially for the overweight and obese. Therefore, the primary objective of this study was to determine the association of BMI with HR_{peak} and with peak oxygen pulse in apparently healthy adults. Differences in the associations in groups of overweight and obese persons compared to normal weight persons were of main interest hereby. The secondary objective was to explore the role of CRF in the association of BMI with HR_{peak}, because CRF is reported to significantly affect the risk of CVD in the overweight and obese.[9–11]

METHODS

Design, setting, participants, and data collection

An observational cross-sectional study with multi-center data originating from the Low-Lands Fitness Registry was done. Use of these data was approved by the Medical Ethics Review Committee (MERC) of the University Medical Center Utrecht under MERC-protocol number: 16-167/C. Data of this registry were collected by CPET in the period from 2000 to 2016, in 11 exercise testing locations (hospitals and sports medicine centers) in the Netherlands. Participants were healthy adults tested for a wide variety of reasons, including fitness checks for physically demanding jobs and intensive sports participation. To be eligible to participate in the Low-Lands Fitness Registry, a subject had to be 18 years or older and apparently healthy. Subjects were excluded from the registry if they used β-blockers. Also, subjects with a present diagnosis or a history with a diagnosis of any cardiovascular disease, pulmonary disease or any other physical or mental diagnosis limiting the execution of CPET were not eligible to participate. Data were selected from the database when a participant was maximally tested with CPET, determined by a peak respiratory exchange ratio (RER_{peak}) of 1.00 or more[37]. Data of participants with an absence of peak oxygen uptake measurements were excluded from the study, because these measurements were needed to calculate peak oxygen pulse.

The cardiopulmonary exercise tests were either performed on a stationary bike or on a treadmill.[17] All tests started with an initial warming-up at very low load or no resistance, and then resistance was increased every minute by various amounts.[17] On the bike resistance was increased with 10 to 25 watts per minute, depending on the used protocol in the testing

center. On the treadmill ramp protocols were used with increased gradients of incline at fixed time intervals. All patients were actively encouraged to continue until exhaustion.

Variables

Patient characteristics collected in the Low-Lands Fitness Registry included: age (years), height (cm), weight (kg), gender (male/ female), and smoking status (no/ yes). Height and weight were used to calculate BMI. BMI was adopted in the dataset as continuous variable, and as a categorical variable, dividing the subjects into underweight (BMI < 18.50 kg $\cdot m^{-2}$), normal weight (BMI = 18.50 – 24.99 kg $\cdot m^{-2}$), overweight (BMI = 25.00 – 29.99 kg $\cdot m^{-2}$), and obese (BMI ≥ 30.00 kg $\cdot m^{-2}$).[38] Relevant recorded testing variables were: peak oxygen uptake (VO_{2peak}), peak carbon dioxide output (VCO_{2peak}), peak heart rate (HR_{peak}), and peak work rate (WR_{peak}). Peak oxygen pulse was calculated by dividing VO_{2peak} by heart rate (HR) at time of reaching VO_{2peak} and expressed in mL O_2/ HR. Peak respiratory exchange ratio (RER_{peak}) was determined by dividing VCO_{2peak} by VO_{2peak}.[39]

Sample size

According to previous research, the effect of BMI on HR_{peak} is expected to be small. [20–26] An effect size (f^2) of 0.02 is assumed to be ''small'' for multiple regression.[39] G*Power[40] was used to calculate needed sample size. To detect an association with one predictor and an f^2 of 0.02 a total of 528 observations was needed to achieve a power of 90% assuming an α-level of 0.05 (Linear Multiple Regression: Fixed Model, R^2 deviation from zero).

Statistical analysis

IBM SPSS Statistics Version 24.0 (Armonk, New York)[41] was used for statistical analyses. Missing values were handled with multiple imputation by chained equations when cases of a variable were missing (completely) at random, by running 40 cycles of imputations.[42] In case of missing not at random the value was accepted as missing. Continuous variables were presented with a mean and standard deviation when normally distributed and with a median and interquartile range in case of a non-normal distribution. Categorical parameters were presented with total numbers and percentages.

The associations of BMI with HR_{peak} and peak oxygen pulse were modeled with two separate linear regression analyses, using BMI as continuous independent variable, and HR_{peak} and peak oxygen pulse respectively as dependent variables. Adjustments for age and gender were made by introducing these variables into the analyses. Dichotomous variables Obese (BMI ≥ 30 kg $\cdot m^{-2}$: yes/ no), and Overweight and obese (BMI ≥ 25 kg $\cdot m^{-2}$: yes/ no) were calculated. These were used to define interactions of these weight subgroups with BMI. The interactions were introduced in the models to compare the associations of BMI with HR_{peak} and peak oxygen pulse between obese and non-obese individuals, and between overweight and obese combined versus underweight and normal weight. The associations of BMI with HR_{peak} and peak oxygen pulse were then further analyzed by separate modeling in subgroups

of BMI categories (underweight, normal weight, overweight, obese, and overweight and obese). To study our secondary aim, the role of CRF in the analyzed associations of BMI with HR_{peak} was then explored by introducing VO_{2peak} (in mL O_2/min) in the main models for HR_{peak} and the defined models for analyses of weight subgroups. Partial R^2 from BMI and VO_{2peak} in the models for HR_{peak} and peak oxygen pulse was noted for all analyses performed. Test results with P-values of less than 0.008 were assumed to be statistically significant, based on Sidak correction method of the 0.05 alpha-level to deal with multiple testing. Number of tests ran was six (one analysis in the total sample and five analyses in the BMI subgroups).

RESULTS

Participant characteristics

Participant characteristics are shown in Table 1. One outlier in a case with a HR_{peak} of 1737 was handled as missing value. 2349 cases of smoking status were potentially missing not at random and therefore accepted as missing. Missing data of other variables were found missing (completely) at random based on missing value patterns and therefore imputed. Almost all continuous variables were non-normally distributed. Therefore, continuous variables were presented using median and interquartile range.

Table 1. Participant characteristics.

Characteristics	Total (n = 7990)	Normal weight (n = 4548)	Overweight (n = 2967)	Obese (n = 411)
Age (years)	31.7 (24.6-42.6)	28.3 (22.9-38.6)	35.8 (27.8-45.2)	41.8 (31.5-48.3.0)
Gender				
Male	6698 (83.8%)	3670 (80.7%)	2676 (90.2%)	309 (75.2%)
Weight (kg)	79.8 (72.3-87.9)	74.5 (68.3-79.9)	87.6 (82.4-93.0)	102.5 (95.4-111.0)
Height (cm)	180.9 (175.3-185.8)	180.1 (175.1-185.9)	181.1 (176.2-185.7)	178.5 (172.1-185.0)
BMI (kg/m^2)	24.5 (22.6-26.4)	23.0 (21.7-24.0)	26.6 (25.7-27.8)	31.6 (30.7-33.3)
BMI category				
Underweight	64 (0.8%)			
Normal weight	4548 (56.9%)			
Overweight	2967 (37.1%)			
Obese	411 (5.2%)			
Smoker				
No	4305 (53.9%)	2370 (52.1%)	1685 (56.8%)	216 (52.5%)
Missing	2349 (29.4%)	1405 (30.9%)	777 (26.2%)	140 (34.1%)
HR_{peak} (bpm)	184 (175-191)	186 (177-192)	182 (172-189)	174 (163-184)
Peak O_2 pulse (mL O_2/ HR)	19.4 (16.7-22.0)	19.0 (16.2-21.3)	20.3 (17.6-22.9)	19.7 (15.6-22.6)
VO_{2peak} (mL O_2/min)	3587 (3006-4029)	3532 (2983-3950)	3707 (3146-4156)	3358 (2625 – 4017)
Relative VO_{2peak} (mL O_2/kg/min)	44.42 (38.31-49.92)	47.2 (41.5-52.0)	42.1 (36.3-46.7)	32.78 (25.91 – 38.72)
RER_{peak}	1.16 (1.12-1.20)	1.16 (1.12-1.20)	1.15 (1.12-1.20)	1.14 (1.10-1.18)

Continuous outcomes presented as median (interquartile range). Abbreviations: BMI, body mass index; HR_{peak}, peak heart rate; bpm, beats per minute; Peak O_2 pulse, peak oxygen pulse; HR, heart rate; VO_{2peak}, peak oxygen uptake; RER_{peak}, peak respiratory exchange ratio.

In the total sample of 7990 participants 5.2% was obese. Median age was higher in the obese group (41.8 years) than in the overweight group (35.8 years) and the normal weight group (28.3 years). In the obese group the proportion of males was smaller (75.2%) than in normal weight group (80.7%). Median HR_{peak} was 12 beats per minute (bpm) lower in the obese group and median peak oxygen pulse was 0.7 mL O_2/ HR higher in the obese group, compared to the normal weight group.

The association of BMI with HR_{peak}

No assumptions of linear regression were violated in the analyses. Table 2 shows the results of the modeled associations of BMI with HR_{peak}. In the total sample, BMI was significantly associated with HR_{peak} ($p < 0.001$) and explained 6% of the variance in HR_{peak}. When corrected for age and gender there still was a significant association of BMI ($p < 0.001$) with HR_{peak}. BMI accounted for 0.5% of the variance of HR_{peak} in this model. Based on the studied interactions (Table 6, appendix), the association between BMI and HR_{peak} was found to be significantly different in the obese compared to the non-obese ($p = 0.001$), but it was not between normal weight versus overweight and obese combined ($p = 0.890$).

Table 2. Associations of BMI and VO_{2peak} with HR_{peak} in the total sample.

Model	Unstandardized β	Standard Error	p	Adjusted R^2
Intercept	207.184	1.144	<0.001	0.060
BMI	-1.037	0.046	<0.001	
Intercept	213.704	0.906	<0.001	0.424
BMI[1]	-0.301	0.038	<0.001	
Age	-0.699	0.011	<0.001	
Female gender	-5.999	0.311	<0.001	
Intercept	199.214	1.085	<0.001	0.459
BMI	-0.441	0.037	<0.001	
Age	-0.623	0.011	<0.001	
Female gender	-0.939	0.375	0.012	
VO_{2peak}[2]	0.004	0.000	<0.001	

Partial adjusted R^2 = [1]0.005; [2]0.035. Abbreviations: BMI, body mass index; VO_{2peak}, peak oxygen uptake (mL O_2/min).

Subgroups of underweight, normal weight, overweight, and obesity, as well as the combined subgroup of overweight and obese, were separately analyzed to study differences in associations of BMI with HR_{peak}. Results of the linear regressions are shown in Table 3. In none of the BMI subgroups the association of BMI with HR_{peak} was significant. In the group of overweight and obese combined the association of BMI with HR_{peak} was significant. Partial adjusted R^2 of BMI in this model was 0.6%.

The association of BMI with peak oxygen pulse

Table 4 shows the results of the modeled associations of BMI with peak oxygen pulse in the total sample. All assumptions of linear regression were met. Corrected for age and gender, BMI was significantly associated ($p < 0.001$) with peak oxygen pulse. The addition of gender

to the univariate model with BMI led to an increase in adjusted R^2 from 0.033 to 0.360. BMI accounted for 2.3% of the variance in peak oxygen pulse in the final model (including BMI, age and gender; total R^2 = 0.366) without interactions. Based on the studied interactions (Table 7, appendix), the association between BMI and peak oxygen pulse was significantly different in the obese compared to the non-obese (p < 0.001) but not between the normal weight and the overweight and obese combined (p = 0.029).

Table 3. Associations of BMI with HR_{peak} in weight subgroups.

Model	Unstandardized B	Standard Error	p	Adjusted R^2
Underweight				
Intercept	213.671	30.951	<0.001	0.587
BMI[1]	-0.414	1.720	0.811	
Age	-0.648	0.113	<0.001	
Female gender	-11.288	1.720	<0.001	
Normal weight				
Intercept	208.150	2.137	<0.001	0.405
BMI[2]	-0.112	0.096	0.244	
Age	-0.664	0.013	<0.001	
Female gender	-5.222	0.364	<0.001	
Overweight				
Intercept	216.583	4.011	<0.001	0.391
BMI[3]	-0.336	0.150	0.025	
Age	-0.743	0.018	<0.001	
Female gender	-7.103	0.663	<0.001	
Obese				
Intercept	220.384	7.006	<0.001	0.380
BMI[4]	-0.368	0.215	0.088	
Age	-0.842	0.058	<0.001	
Female gender	-5.732	1.526	<0.001	
Overweight and obese				
Intercept	219.653	2.138	<0.001	0.404
BMI[5]	-0.436	0.077	<0.001	
Age	-0.755	0.017	<0.001	
Female gender	-6.835	0.600	<0.001	

Partial adjusted R^2 = [1]0.000; [2]0.000; [3]0.001; [4]0.003; [5]0.006. Abbreviations: BMI, body mass index.

Table 4. Association of BMI with peak oxygen pulse in the total sample.

Model	Unstandardized β	Standard Error	p	Adjusted R^2
Intercept	13.354	0.358	<0.001	0.033
BMI	0.239	0.014	<0.001	
Intercept	16.105	0.293	<0.001	0.366
BMI[1]	0.210	0.012	<0.001	
Age	-0.030	0.003	<0.001	
Female gender	-6.343	0.101	<0.001	

[1]Partial adjusted R^2 = 0.023. Abbreviations: BMI, body mass index.

Further analyses of weight subgroups showed a significant association of BMI with peak oxygen pulse (p < 0.001) in the normal weight and in the obese groups (Table 5). In these

models BMI explained 3.3% of the variance in peak oxygen pulse in the normal weight sample and 1.6% in the obese. In the underweight and overweight subgroup BMI was not significantly associated with peak oxygen pulse. In the subgroup of overweight and obese individuals combined a significant association of BMI with HR_{peak} (partial R^2 = 0.003) was found as well.

Table 5. Association of BMI with peak oxygen pulse in weight subgroups.

Model	Unstandardized β	Standard Error	p	Adjusted R^2
Underweight				
Intercept	12.548	9.481	0.191	0.275
BMI[1]	0.126	0.026	0.811	
Age	0.049	0.035	0.163	
Female gender	-3.709	0.722	<0.001	
Normal weight				
Intercept	9.018	0.708	<0.001	0.383
BMI[2]	0.492	0.032	<0.001	
Age	-0.011	0.004	0.015	
Female gender	-5.660	0.121	<0.001	
Overweight				
Intercept	21.402	1.258	<0.001	0.316
BMI[3]	0.065	0.047	0.166	
Age	-0.063	0.006	<0.001	
Female gender	-7.149	0.208	<0.001	
Obese				
Intercept	18.217	1.789	<0.001	0.536
BMI[4]	0.211	0.055	<0.001	
Age	-0.094	0.015	<0.001	
Female gender	-7.983	0.390	<0.001	
Overweight and obese				
Intercept	20.679	0.652	<0.001	0.354
BMI[5]	0.098	0.024	<0.001	
Age	-0.066	0.005	<0.001	
Female gender	-7.304	0.183	<0.001	

Partial adjusted R^2 = [1]0.000; [2]0.033; [3]0.000; [4]0.016; [5]0.003. Abbreviations: BMI, body mass index.

The role of CRF

As shown in Table 2 and Table 8 (appendix) CRF was found to be significantly associated with HR_{peak} in the total sample and all weight subgroups that were analyzed ($p < 0.001$). CRF accounted for 3.3% or 3.4% of the variance in HR_{peak} in all subgroups except from the obese. In obese individuals partial adjusted R^2 was 5.3%. In the subgroups of overweight and obese female gender was no longer significantly associated with HR_{peak} after the introduction of CRF to the models.

DISCUSSION

Primary aim of this study was to determine the association of BMI with HR_{peak} and peak oxygen pulse and to compare the associations in the overweight and obese with the normal weight and underweight. Significant associations were found of BMI with both parameters of cardiac performance during exercise and these differed between normal weight and obese persons. However, the impact of BMI was very low and therefore the associations of BMI with HR_{peak} and peak oxygen pulse can be assumed to be of low clinical relevance.

Several further analyses were performed in weight subgroups (underweight, normal weight, overweight, and obese) and revealed no significant associations. In the subgroup of overweight and obese combined BMI was found to be significantly associated with HR_{peak}, though the variance in HR_{peak} explained by BMI was very low as it was in the total sample as well. Unstandardized betas were also low: compared to the normal weight, predicted HR_{peak} was only 4 bpm lower when BMI increased with 10 kg $\cdot m^{-2}$ for example. A higher BMI has therefore very limited impact on predicted HR_{peak} values.

Our results can be compared to the outcomes of previous research. In contrast to our study almost all studies primarily focused on the association of age with HR_{peak} though. BMI accounted for 0.5% of the variance in HR_{peak} in our main model (Table 2). Gondoni et al. reported a higher partial R^2 value of 5.8%.[18] Gellish et al. found a non-significant β of BMI of -0.07.[20] Maximum BMI in their sample was 42.9 kg/m^2, but it is unclear what the proportion of overweight and obese persons was. The proportion of overweight and obese persons in the study by Nes et al. was 68%.[21] Adding BMI categories to their model led to a negligible change in R^2. They did not find a significant difference in association of age with HR_{peak} across BMI categories. This is contradictory with the outcomes of the study by Zhu et al. who found a significantly greater decline in HR_{peak} predicted by age in the highest BMI category (overweight and obese) in their cohort compared to a lower BMI.[24] The results of our study showed a comparable effect, but this was very small and therefore irrelevant for clinical decision making. It should be noted that Nes et al. and Zhu et al. did not study BMI as a continuous predicting parameter as we did.[21,24] Across the board, research seems to agree that the impact of BMI in predicting HR_{peak} is very low. We confirmed this in our study and demonstrated that differences of this effect in the overweight and obese compared to the normal weight are very limited.

Of the parameters in our study, gender was, as expected, found to be of main influence on peak oxygen pulse. Predicted peak oxygen pulse differed 6.343 mL O_2/HR between males and females. BMI was also significantly associated with peak oxygen pulse. Unstandardized β of BMI in the model was 0.210, thus oxygen pulse is higher at maximal exertion in persons with a higher BMI. HR_{peak} is slightly lower though in persons with a higher BMI, as found in our study. To achieve the reported higher levels of oxygen transport per heartbeat, the heart needs to work harder by increasing stroke volume, to compensate for a lower HR_{peak} in

overweight and obese individuals. During exercise the heart is therefore put at higher stress in these BMI groups. This could lead to left ventricular hypertrophy eventually, which is an independent risk factor for heart failure[43], and is in line with previous research reporting maximal cardiac output to be higher in obesity with a larger stroke volume.[19]

The heart is pumping higher blood volumes per minute in obese individuals, but absolute VO_{2peak} was only slightly different between obese and non-obese. An explanation for this might be an underlying cardiovascular dysfunction presented by a lower arteriovenous oxygen difference.[19] Cardiac response at maximal exercise is thus compromised in obese individuals compared to normal weight.[14] This was reflected in the study by Koch et al. on reference standards for CPET in healthy adults, who observed divergent CPET parameters (VO_{2peak} as absolute value and relative to body weight) in obese adults.[44] They found obesity to significantly influence the studied CPET outcomes as a confounder and therefore excluded obese individuals from their study. HR increases faster and seems to reach its peak value earlier in obese individuals. This might be caused by an impaired response of the sympathetic system at maximal exercise levels.[18] In our study, adding VO_{2peak} to the models led to a significant increase of explained variance in HR_{peak}. This also had impact on the unstandardized β's of BMI. These increased, with as a consequence a bigger effect on HR_{peak} in individuals with a lower VO_{2peak} and thus a lower CRF. The impact on HR_{peak} was still low though, so CRF is not seriously affecting prediction models to determine training intensity based on HR_{peak} values in overweight and obese individuals. Cardiac response during exercise might however be more compromised in the unfit overweight and obese individuals than in the fit. This might be another element in the intervening role of CRF in the association of overweight and obesity with CVD.[9–11]

The following strengths were recognized. We were able to study associations in a large sample and in large BMI subgroups which strengthened the statistical power with the tests. Furthermore, observational testing data from regular exercise tests were used, limiting the risk for selection bias. Our study also had some limitations that are important to discuss. Data on duration of obesity were not available from the Low-Lands Fitness Registry. This factor could play a major role in morphological changes in the heart and its performance during exercise, mainly caused by left ventricular morphology.[45] Ventricular function is altered in the overweight and obese, with left ventricular hypertrophy significantly influencing HR_{peak}.[30,45–47] Unfortunately, no echo or MRI data on ventricular function were available to us. Because left ventricular dysfunction develops with time, duration of overweight and obesity might have influenced the found association of BMI with HR_{peak} and peak oxygen pulse in our study and could be a topic of future research.[43] Second limitation concerns the demographic composition of the study sample. Median age in the cohort was around 30 years old, with a right skewed distribution. Older individuals were underrepresented, males and females were unevenly represented, and participants were recruited from the Dutch population, so the sample lacked racial diversity. These factors might limit the generalizability of the reported results. Last limitation to discuss is the use of absolute VO_{2peak} in the secondary analyses.

Using absolute VO_{2peak} as measure of CRF in obese individuals is arguable, because it might overestimate the real fitness in obesity. We have chosen to do so however, because the factor body weight was already present in the models by the parameter BMI. Furthermore, VO_{2peak} relative to total body mass is found to overestimate CRF in obesity as well.[48,49] VO_{2peak} relative to lean body mass is a better representation of CRF in obese individuals[50], but data on fat free mass was not routinely included in the Low-Lands Fitness Registry.

Taken all the discussed considerations into account, the results of our study suggest that BMI is not a factor to consider in exercise prescription in overweight and obese individuals. Also, dealing with BMI as a confounder in HR_{peak} models as several studies did, seems to be unnecessary. The found associations of BMI with HR_{peak} and peak oxygen pulse do not seem to be of clinical relevance and we argue that no future research is needed to further study these associations. CRF plays an intervening role in the association of BMI with HR_{peak}, but the impact on HR_{peak} is limited and might not be relevant in determining training intensity based on predicted HR_{peak} values.

CONCLUSION

BMI is significantly associated with HR_{peak} and peak oxygen pulse and these associations differ between overweight and obese individuals compared to individuals with a normal body weight. The impact of BMI on HR_{peak} and peak oxygen pulse is limited and may not be relevant for clinical decision making. CRF plays an intervening role in the association of BMI with HR_{peak}, but clinical implications are limited.

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APPENDIX

Table 6. Association of BMI with HR_{peak} with modeled interactions.

Model	Unstandardized β	Standard Error	p	Adjusted R^2
Intercept	211.493	1.119	<0.001	0.424
BMI	-0.204	0.048	<0.001	
Age	-0.702	0.011	<0.001	
Female gender	-5.842	0.315	<0.001	
BMI*Obese	-0.067	0.020	0.001	
Intercept	213.856	1.423	<0.001	0.424
BMI	-0.308	0.064	<0.001	
Age	-0.699	0.011	<0.001	
Female gender	-5.997	0.312	<0.001	
BMI*Overweight and Obese	0.002	0.014	0.890	

Abbreviations: BMI, body mass index.

Table 7. Association of BMI with peak oxygen pulse with modeled interactions.

Model	Unstandardized β	Standard Error	p	Adjusted R^2
Intercept	14.535	0.361	<0.001	0.370
BMI	0.279	0.015	<0.001	
Age	-0.032	0.003	<0.001	
Female gender	-6.232	0.102	<0.001	
BMI*Obese	-0.047	0.006	<0.001	
Intercept	15.331	0.461	<0.001	0.367
BMI	0.246	0.021	<0.001	
Age	-0.029	0.003	<0.001	
Female gender	-6.353	0.101	<0.001	
BMI*Overweight and Obese	-0.010	0.005	0.029	

Abbreviations: BMI, body mass index.

Table 8. Associations of BMI and VO_{2peak} with HR_{peak} in weight subgroups.

Model	Unstandardized B	Standard Error	p	Adjusted R^2
Underweight				
Intercept	202.885	30.540	<0.001	0.609
BMI	-0.584	1.675	0.729	
Age	-0.639	0.110	<0.001	
Female gender	-7.656	2.872	0.010	
VO_{2peak}[1]	0.004	0.002	0.040	
Normal weight				
Intercept	200.087	2.136	<0.001	0.437
BMI	-0.449	0.096	<0.001	
Age	-0.610	0.013	<0.001	
Female gender	-1.041	0.438	0.018	
VO_{2peak}[2]	0.004	0.000	<0.001	
Overweight				
Intercept	198.054	4.151	<0.001	0.424
BMI	-0.365	0.146	0.012	
Age	-0.638	0.019	<0.001	
Female gender	-1.470	0.776	0.058	
VO_{2peak}[3]	0.004	0.000	<0.001	
Obese				
Intercept	194.114	7.899	<0.001	0.433
BMI	-0.557	0.208	0.008	
Age	-0.634	0.065	<0.001	
Female gender	3.712	2.097	0.077	
VO_{2peak}[4]	0.006	0.001	<0.001	
Overweight and obese				
Intercept	200.022	2.477	<0.001	0.438
BMI	-0.478	0.075	<0.001	
Age	-0.639	0.019	<0.001	
Female gender	-0.742	0.718	0.301	
VO_{2peak}[5]	0.004	0.000	<0.001	

Partial adjusted R^2 = [1]0.022; [2]0.032; [3]0.033; [4]0.006; [5]0.034. Abbreviations: BMI, body mass index, VO_{2peak}, peak oxygen uptake (mL O_2/min).

SAMENVATTING

De associatie van BMI met piekhartslag en piek zuurstofpuls bij ogenschijnlijk gezonde volwassenen.

Achtergrond: verbeteren van de cardiovasculaire fitheid (CVF) is belangrijk om risico's op gezondheidsproblemen te verkleinen. Om effectief te kunnen trainen en CVF te verbeteren is het van belang te weten hoe het hart reageert op inspanning. De hartfunctie is afwijkend bij mensen met obesitas vergeleken met mensen met een normaal lichaamsgewicht. De associatie tussen de body mass index (BMI) en de piekhartslag (HR_{piek}) en piek zuurstofpuls tijdens maximale inspanning is niet goed bekend.

Doelstelling: primaire doel was het bepalen van de associatie tussen BMI en de HR_{piek} en de piek zuurstofpuls bij ogenschijnlijk gezonde volwassenen. We waren daarbij geïnteresseerd in verschillen in deze associaties tussen mensen met een normaal lichaamsgewicht en mensen met overgewicht en obesitas. Tweede doel van het onderzoek was de rol van CVF in de associatie tussen BMI en HR_{piek} te exploreren.

Methode: een cross-sectionele observationele studie werd uitgevoerd bij ogenschijnlijk gezonde volwassenen. Data met uitslagen van inspanningstesten uit de Low-Lands Fitness Registry werden gebruikt om de associatie van BMI met HR_{piek} en piek zuurstofpuls te analyseren met een lineaire regressieanalyse. Subanalyses werden uitgevoerd om verschillen in deze associaties tussen subgroepen van gewichtsklassen te onderzoeken en de rol van CVF werd onderzocht door deze variabele toe te voegen aan het regressiemodel.

Resultaten: er werden 7990 participanten geïncludeerd. Mediane leeftijd was 31.7 en 5.2% was obees. Er werd een significante associatie gevonden tussen BMI en HR_{piek} in de gehele onderzoekspopulatie, maar niet in subgroepen van gewichtsklassen. Op basis van geanalyseerde interacties werd een significant verschil gevonden van de associatie van BMI met HR_{piek} tussen mensen met en zonder obesitas. De associatie tussen BMI en piek zuurstofpuls was ook significant in de gehele onderzoekspopulatie en in enkele gewichtsklassen. Partiële verklaarde variantie van BMI in de regressiemodellen was 0.000 – 0.033.

Conclusie: BMI is geassocieerd met HR_{piek} en piek zuurstofpuls en deze associaties verschillen tussen mensen met en zonder obesitas. CVF beïnvloedt de associatie tussen BMI en HR_{piek}.

Klinische relevantie: de impact van BMI op HR_{piek} en piek zuurstofpuls is beperkt en mogelijk niet relevant voor klinische besluitvorming.

Steekwoorden: body mass index; piek hartslag; piek zuurstofpuls; obesitas; cardiovasculaire fitheid.